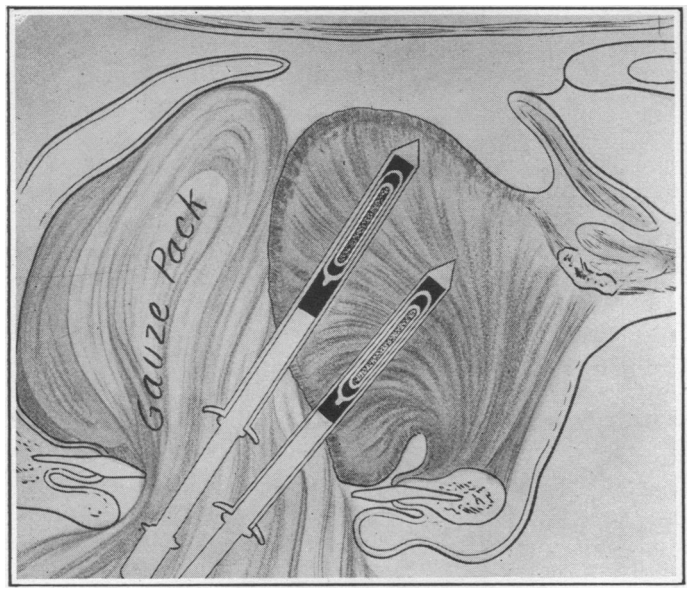

Fig. 12.—Trocars have been converted into containers for 50 milligram tubes of radium which are driven into the coagulated half of the tongue near its middle. Mouth is packed with gauze and patient kept under morphin while radium is in place.

to the superiority of an electric knife or cautery knife over a steel knife, I have not been fully convinced. It seems reasonable that if we cut entirely outside the cancer it really does not matter what kind of a knife we use. The patient will be cured anyway. On the other hand, if we cut through a cancer, it does not matter what kind of a knife we use; the patient will not be cured anyway. Those more skilled with an electric knife or cautery knife may do better work with these agencies. Good surgeons who are not skilled with these agencies will probably do better with the ordinary knife. One skilled in both methods will use both knives, to suit the case in hand.

The one outstanding field for electrocoagulation is in the mouth, particularly in cancer of the tongue. By passing a loop of thread or silkworm through the tongue, it may be pulled out and easily anesthetized. Let us consider a cancer located midway between the tip and base of the tongue on one side. The tip of the tongue is first anesthetized around the traction loop. The tongue is then drawn out. The left index finger is passed down to the base of the tongue. With a long needle, the tongue is fully infiltrated with novocain solution on the side of the raphe corresponding to the growth. Long needles are then inserted in the tongue near the raphe and the one farthest back is pushed almost to the surface of the root of the tongue, the finger acting as a guide to avoid injury to the epiglottis. The coagulation current is made to contact the various needles until the entire side of the tongue is cooked and turns black. Two fifty milligram tubes of radium, enclosed in silver capsule and put in large trocars in which a trocar point closes the end, are driven into the dead side of the tongue, following the holes from which the coagulation needles have been removed. The lower or more remote trocar containing radium is driven through the length of the tongue, well down to the base but not puncturing the mucous membrane. The other trocar stops somewhat nearer the tip. If the floor of the mouth is involved with the cancer, extra radium needles are driven into this area. While the tongue is pulled out, a large pack of gauze is made to fill the entire mouth, literally stuffing the mouth, including the buccal cavity. The ends of the radium containers are brought out through the mouth. The teeth are held apart with the gauze, and radium containers are firmly fixed by the gauze pack. Patient is then given morphin in doses sufficient to keep him entirely comfortable. The radium may be left in for twenty-four hours. We have used as much as four thousand milligram hour doses in this way at one treatment. If properly packed, there is no injury to the other mucous membrane and the treatment is most thorough. A few days later the dead tongue may be trimmed away. The patient has one-half of the tongue left, which has been thoroughly radidated (Figs. 9, 10, 11, and 12). We have tried no other method of treatment of cancer of the tongue that is comparable to this combination.

611 Lovejoy Street.

CHRONIC NONVALVULAR HEART DISEASE—ITS CAUSES, DIAGNOSIS, AND MANAGEMENT*

By HENRY A. CHRISTIAN, M. D.
Boston, Massachusetts

AMONG adults this is the form of chronic cardiac disease encountered most frequently, comprising, in my clinic at the Peter Bent Brigham Hospital, 61 per cent of the patients diagnosed as having some form of chronic heart disease in a ten-year period. By chronic nonvalvular heart disease we understand that form of cardiac failure in which the defective function is due to myocardial disturbance, for in these patients valves and pericardium show no organic lesion. Usually there is cardiac hypertrophy and dilatation; rarely there is an interstitial (fibrous) myocarditis; there may be coronary sclerosis, but in very many of these patients the arteries of the myocardium are normal. Microscopically there may be evidences of degenerative changes in the muscle fibers, but in the majority of cases the microscope reveals no change other than hypertrophy of the fibers. Occasional foci of round-cell infiltration and scattered areas of fibrosis may occur. Generalized fibrosis rarely is found.

It would seem as if we had, in these patients, the paradox of a powerful, healthy looking heart

* Annual Scripps Metabolic Clinic Lecture before the San Diego County Medical Society at La Jolla, California, January 25, 1930.

muscle, which actually was unable to carry on the ordinary circulatory function needed in daily life. It does seem to be a fact that once the heart has enlarged, it has already begun on a career of increasing inefficiency. Four years ago, in an address before the Southern Medical Association, I stated it in this way: "It seems to be a clinical fact that, so soon as a heart begins to enlarge, it has commenced a cycle of changes that, in a relatively short time, will result in signs of some circulatory disability." Within a few weeks Cloetta (*Journal of the American Medical Association,* November 9, 1929), has expressed the same idea, saying: "Contrary to the former conception, I now consider every heart with dilatation and hypertrophy as in an abnormal state and of diminished efficiency."

CAUSES

If heart hypertrophy is a malevolent rather than a benevolent process, the real problem then is, what causes the heart to hypertrophy? Various explanations have been offered. A popular explanation is that it is a work hypertrophy, incident usually to hypertension. Some observers go so far as to say that in all patients of this group there has been, at some time, a maintained hypertension, even if blood pressure is normal when the patient is observed after cardiac failure has begun. I believe, however, that observations have been made over long enough periods prior to cardiac disturbance in enough patients to justify not agreeing that hypertension has been a cause of this change in all cases. In many of the patients, however, it has existed. However, some other causative factor must enter, for so often we observe patients who have sustained hypertension for long periods without cardiac hypertrophy as well as those with cardiac hypertrophy and no hypertension. Furthermore in such individuals as long-distance runners, who place an increased amount of work on their circulation, there is no cardiac hypertrophy or dilatation. The heart actually grows smaller during a twenty-five-mile run in a successful long-distance runner.

Arteriosclerosis and syphilis are not present often enough to be important causative factors. Rheumatic fever has not occurred. Infections at times seem to have a very direct relationship, but various infectious diseases and focal infections seem no more frequent in this type of cardiac disease than in similar control groups with no cardiac disease. Valve lesions do not occur, except dilatation of the valve ring after the process is well advanced. Arrhythmias appear too late to be of any significant causative effect.

In other words, no common antecedent condition can be discovered in studying the past history of these patients to account for the development of the cardiac disturbance.

There are experimental studies indicating that strain of not long duration may lead to subsequent hypertrophy and dilatation of the heart. Strain, combined with varying other factors, such as hypertension, infection, etc., may play an important rôle in etiology.

It is probable that the relationship of the cardiac musculature to a blood-containing cavity, as in the heart, may determine a difference between the response of cardiac and skeletal muscle to increased demands on their function. In the heart, in order to increase cardiac output, cardiac cavities must dilate. This stretches the muscle wall. Increased tension on muscle fibers is known to increase the amount of work a muscle can do. If hypertrophy is a response to this physiologic process, then the cavity enlarges more and this repeats itself in cycles. As Cloetta has put this, "It must not be forgotten, however, that the heart is a spherical organ and that it contracts around a fluid content. No sooner does the heart dilate than this fluid mass or resistance increases, thereby partly nullifying the advantage gained by dilatation. Thus a stage must be reached at which improvement due to cardiac hypertrophy is overcompensated by the greater load, and this is where cardiac insufficiency really begins; the heart has stretched itself beyond its physiologic limits and signs of insufficiency, such as diminished volume of beats and lessened capacity, begin to appear." This seems to be what happens.

DIAGNOSIS

Diagnosis of this type of cardiac failure is not difficult. There are the usual evidences of cardiac insufficiency. There are the physical signs of cardiac enlargement, for it is very rare for the heart not to be enlarged. Evidences of valve lesion are lacking. There is no history of rheumatic fever. Most of the patients are past forty. A systolic murmur may be heard or there may be no murmurs. Rhythm often is regular, but there may be extrasystoles or auricular fibrillation. Other arrhythmias occur but are unusual.

Two groups of these cases are misdiagnosed with considerable frequency: (1) The markedly edematous patient with a regular, not very rapid, pulse often is considered as a case of nephritis with edema. The urine, containing albumin and casts, suggests nephritis, but the urine picture is due to passive congestion of the kidney, as shown by the speedy disappearance of albumin and casts as a sequence to adequate cardiac therapy. (2) The patient with paroxysmal type of dyspnea is regarded as having bronchial asthma or asthmatic bronchitis, the underlying cardiac disturbance having been overlooked, in part due to the increased difficulty in making out the enlargement of the heart owing to pulmonary emphysema, and in part owing to the physical signs of chronic bronchitis so often seen in patients of this type.

These two diagnostic mistakes are of more than academic interest because, if the cardiac disturbance is not recognized, the patients are given treatment appropriate to the erroneous diagnosis of nephritis or asthma and fail to respond, whereas if treated as patients with cardiac failure the response often is dramatically successful.

TREATMENT

Treatment for these patients is that for other forms of cardiac failure, with rest in bed, diet, digitalis, etc., in adequate dosage. In the ones

with more marked edema diuretics may be required to remove the edema though very frequently the digitalis is all that is needed. Any form of potent digitalis is satisfactory, and any method of dosage may be followed. The important thing is to give sufficient digitalis to produce a digitalis action.

It is a common error to believe that in this group of patients digitalis has little effect in the absence of auricular fibrillation. It is true that digitalis produces marked therapeutic effects in patients with auricular fibrillation. It is equally true that it is just as effective in patients with regular rhythm. In the two groups of patients already mentioned as frequently misdiagnosed as nephritis with edema or bronchial asthma respectively, digitalis therapy often gives brilliant effects. What can be more dramatic than the rapid disappearance of excessive edema or the cessation of paroxysms of severe dyspnea, as one so often sees follow adequate digitalis therapy in these patients?

In patients with chronic nonvalvular cardiac disease one often sees great benefit from a daily ration of digitalis, 0.1 to 0.15 gram of powdered digitalis leaves per twenty-four hours, or corresponding amounts of other digitalis preparations, kept up long after all obvious evidences of cardiac insufficiency have disappeared. As I watch these patients I am becoming more and more convinced of the value of this form of usage of digitalis and inclining more and more to use these daily doses of digitalis in patients with cardiac hypertrophy even before there develop any very evident signs of decompensation.

Digitalis is a drug peculiarly well adapted to give a continued effect from interval doses. The pharmacologists have taught us the underlying principles responsible for this. We have learned that as digitalis circulates through the heart muscle, it passes through the vessel wall to become fixed in the heart muscle, where it is inactive until it is split up into an active form, a toxigenin or aglykon. This splitting up goes on gradually and the split-product produces the digitalis effect. Straub and Cloetta have been particularly active investigators in this matter. This is the process that permits of continued digitalis effect at a reasonably steady rate without any toxic effects. If the amount of digitalis given in a single dose is increased beyond a certain point, then this new digitalis is fixed in the muscle before that previously there has been split up completely and has finished producing its digitalis action. So cumulative and toxic effects appear.

There is experimental work to indicate that continued use of small doses of digitalis in animals with damaged aortic valves retards cardiac hypertrophy. If so, then there is additional reason for giving digitalis in daily rations in the early stages of the development of those cardiac lesions now under discussion, for, as we have already seen, hypertrophy in itself, as it increases, is a detrimental process. These experiments are in accord with certain clinical observation of the benefit from continued use of small doses of digitalis.

Diuretics are very valuable drugs to remove excessive edema not satisfactorily decreasing from digitalis alone. Diuretics should be given at the time digitalization has been obtained. Diuretics are more satisfactory in their results when given in one or several doses before noon and not repeated on the next or second succeeding day. Of them I have found theobromin sodiosalicylate (diuretin), 0.5 gram by mouth at 8, 10, and 12 o'clock; theophyllin (theocin), 0.3 gram the same way; novasurol (merbaphen) and salyrgan (both given as one dose early in the day, preferably intravenously) most useful and effective, as a rule, in the order in which I have named them. Of these the first two have the advantage of effectiveness by mouth dosage, while the second two require intramuscular or intravenous routes of administration, preferably the latter, as they are somewhat irritating even after deep injection. The second two act better after a preliminary period of three to four days on which the patient receives from three to four doses of one gram of ammonium chlorid or ammonium nitrate. Larger doses of these, as often recommended, may cause nausea and, in my experience, give no better results than the one-gram doses just advised. If there is gastric upset, the ammonium chlorid may be given by rectum.

Treatment along the general lines, as just described, gives very satisfactory results, and often it is most surprising how much may be accomplished in patients apparently in very bad condition. The skillful combination of these methods to obtain such results is the evidence of that sound clinical knowledge which our patients should expect of us.

Peter Bent Brigham Hospital.

EPIDEMIC CEREBROSPINAL FEVER ON THE PACIFIC COAST*

By J. C. GEIGER, M. D.
San Francisco

EPIDEMIC cerebrospinal fever occurs with piquant irregularity. Indeed, since the outbreak of 1904-05, this disease has been sometimes epidemic, sometimes sporadic, without complete cessation. Europe, Africa, South America, Australia, and China have been harassed.

EPIDEMIOLOGY

In other words, the disease has been pandemic practically over the world between 1904-10 with never a real quiescent period in the United States or Europe. In fact, in the United States, each winter, in one locality or another, groups of cases have occurred. There is no doubt that severe epidemics leave viable foci which add to the continuity of the propagation of the disease. The meningococcus only survives in nature in the human being. The epidemiology is by no means as simple as it seems. The epidemiology of pneu-

* Read before the Pacific Interurban Clinical Club, San Francisco meeting, December 19 and 20, 1929.